Invited commentary

Sleep duration and cardiometabolic risk  Commentary on Abe et al., “Sleep duration is significantly associated with carotid artery atherosclerosis incidence in a Japanese population”

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Dozens of studies, spanning over 40 years and multiple continents, have reliably documented that self-reported short and long sleep durations are associated with increased mortality risk [1,2]. A recent meta-analysis [3] reports that the pooled relative risk (RR) of all-cause mortality was RR = 1.10 for short sleep duration and RR = 1.23 for long sleep duration. Specific cardiovascular mortality was reported to be RR = 1.06 for short sleep and RR = 1.38 for long sleep.

Epidemiologic studies of habitual short sleep duration have found that sleep <6 h is associated with abnormalities in blood pressure, lipoproteins, glucose regulation, metabolic hormones, and obesity [4,5]. Less well-studied has been long sleep duration. However, many studies in the past two decades have found that individuals who report long sleep duration are at risk for a number of negative health outcomes as well [2,6]. Associations with long sleep duration have been reported for depression [7], cardiovascular disease [8], stroke [9], hypertension [10], diabetes [11], obesity [12], metabolic syndrome [13], dyslipidemia [14], and a number of cardiometabolic risk factors [2,6].

The study by Abe and colleagues in this issue represents one of the first to carefully assess atherosclerosis relative to sleep duration. This study, of >2000 residents of a Japanese community consisting of individuals 40–84 years of examined intima-medial thickness (IMT), a marker of carotid artery arteriosclerosis. Although the sample was rather small for an epidemiologic study, the authors found that mean IMT increased significantly as self-reported sleep duration increased from 6 to 7 to 8 to ≥9 h of sleep. Moreover, those with longer sleep durations were more likely to exhibit IMT ≥1.2 mm, which is a clinically accepted marker of atherosclerosis. The relationship of sleep duration with IMT was maintained even after controlling for a wide range of possible confounders, including sociodemographics, substance use, and other cardiometabolic measures. These data are consistent with other epidemiologic evidence linking long sleep with a higher risk of heart disease and stroke. Although other cardiovascular and metabolic markers were measured, including blood pressure, cholesterol, insulin, glucose, and others, the present study did not evaluate whether these also varied by sleep duration.

This study is consistent with other epidemiologic studies indicating that risks of long sleep can be greater than risks associated with short sleep. Further, these studies demonstrate that the prevalence of long sleep (>8 h) may be greater than that of short sleep <6 h. However, whereas such epidemiologic data has been an impetus for a dramatic increase in research exploring health effects of insufficient sleep, there has been far less investigation regarding potential hazardous effects of long sleep. Nonetheless, accumulating evidence from approximately 100 studies provides justification for expecting that sleep, like perhaps all other health-related behavior, is indeed hazardous in excessive amounts.

As Abe et al. note, epidemiologic studies cannot establish causality. The associations of long sleep with mortality/morbidity could be confounded by existing morbidities (as could the links with short sleep). However, the analyses of Abe and colleagues (and many other studies) have controlled for these factors to varying degrees. Moreover, a recent prospective study by Mesas et al. [15] found that the association of long sleep with mortality was equally apparent in older adults who had very good physical and psychological health at baseline. Furthermore, other recent studies show that the risks of...
long sleep are equally apparent when sleep is assessed objectively [16–18].

Collectively, the findings by Abe et al. and others regarding sleep duration exist in the larger context of findings that demonstrate that insufficient or excessive sleep duration and/or poor sleep quality (e.g., sleep fragmentation, sleep initiation difficulties) play an important role in several domains of health relevant to cardiometabolic outcomes, including obesity, cardiovascular disease, metabolic dysregulation, and immune dysfunction.

As existing evidence from epidemiological and experimental domains elucidates particular pathways by which sleep impacts health, and how these pathways may interact (i.e., downstream effects of sleep duration and quality), increased attention will need to be paid to the determinants of sleep (i.e., upstream factors). After all, if sleep attainment is crucial to health and longevity, a more complete understanding of how an individual’s sleep is determined is necessary. As with other aspects of health driven by behavior (such as diet and physical activity), an individual’s sleep attainment is an expression of factors at the individual level (e.g., demographics, genetics, behavior), which is embedded within a community level (e.g., neighborhood, culture, family), which itself exists within the wider context of societal factors which could influence sleep (e.g., public policy, exposure to electronic media, working around the clock). These factors, collectively, need to be better understood in their role as determinants of sleep duration and quality.

When the (upstream) determinants of sleep physiology and sleep-related behaviors become better understood, and this knowledge is complemented by a better understanding of the (downstream) consequences of insufficient, excessive or otherwise poor quality sleep, we will better be able to view sleep in the context of health and society. Using this knowledge, there is the potential to develop targeted interventions at the individual, community and societal level, aimed at improving the sleep, and, consequently, the health and longevity, of the population.

References